ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

Research progress on the correlation between plasma cluster protein levels and cognitive dysfunction in patients with Parkinson's disease

Ayinishahan Maimat[1], Ma Jianhua[1,*]

[1]Department of Neurology, The First Affiliated Hospital of Xinjiang Medical University, Urumqi, Xinjiang Uygur Autonomous Region, 830002, China
2543506663@qq.com
[*]Corresponding author

Abstract: *This review focuses on the research progress on the correlation between plasma cluster protein levels and cognitive dysfunction in patients with Parkinson's disease. As a molecule with multiple physiological functions, plasma cluster protein has attracted much attention in the study of cognitive dysfunction in Parkinson's disease, and its role in cell protection and protein metabolism may be related to the pathophysiological process of Parkinson's disease on the basis of elaborating the multi-dimensional manifestations of cognitive dysfunction in Parkinson's disease and the multi-faceted characteristics of plasma cluster protein. Some studies have shown abnormal changes in plasma cluster protein levels in patients with severe cognitive dysfunction, but the results vary depending on factors such as samples, detection methods, and disease stages. Plasma cluster proteins may also interact with other biomarkers such as α-synuclein, tau protein, etc., to affect cognitive function. However, current studies are limited by small sample sizes, lack of standardization of detection methods, and unclear mechanisms. This study deeply analyzes the limitations of existing research and prospectively plans multiple paths for future research, aiming to provide comprehensive and in-depth knowledge integration and research orientation for the research field related to cognitive dysfunction in Parkinson's disease.*

Keywords: *plasma cluster protein; Parkinson's disease; cognitive dysfunction; Pathogenesis; Treatment strategies*

1. Introduction

Parkinson's disease (PD) is a common neurodegenerative disease that seriously affects motor function and quality of life [1]. In recent years, with the in-depth research on Parkinson's disease, non-motor symptoms, especially cognitive dysfunction, have gradually become a research hotspot in this field. Cognitive dysfunction not only brings a lot of inconvenience to patients' daily life, but also greatly increases the burden on family and society. Therefore, it is of great importance to explore the biomarkers associated with cognitive dysfunction in patients with Parkinson's disease and to understand the underlying pathophysiological mechanisms [2,3].

Plasma cluster proteins (PCPs), as a widely present and functionally diverse molecule in a variety of biological fluids, play a key role in physiological processes such as neuroprotection, lipid transport, complement regulation, and apoptosis [4]. Its unique biology has made it stand out in the study of neurodegenerative diseases. In the complex pathological process of Parkinson's disease, plasma cluster proteins may be associated with cognitive dysfunction through multiple pathways. On the one hand, it may rely on its cell protection function to resist the invasion of harmful factors such as oxidative stress and neuroinflammation faced by neurons, thereby maintaining cognitive function. On the other hand, given its role in protein metabolism, it may be able to intervene in the abnormal folding and aggregation of Parkinson's disease-related proteins, thereby affecting the state of cognitive function [5].

Although some studies have begun to focus on the relationship between plasma cluster protein levels and cognitive dysfunction in patients with Parkinson's disease, there are still many uncertainties and controversies in the current research results. Different studies have shown considerable differences in the trends of plasma cluster protein levels and the specific patterns of their association with cognitive dysfunction [6]. In addition, the detailed mechanisms by which plasma cluster proteins precisely affect

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

cognitive function in the pathological context of Parkinson's disease are still poorly understood. Therefore, it is particularly urgent and crucial to systematically sort out and deeply explore the correlation between plasma cluster protein levels and cognitive dysfunction in patients with Parkinson's disease [7].

2. Clinical manifestations, pathogenesis and pathological causes of cognitive dysfunction in Parkinson's disease

2.1 Clinical manifestations of cognitive dysfunction in Parkinson's disease

The clinical manifestations of cognitive dysfunction in Parkinson's disease are complex. People are easily distracted and have difficulty concentrating when reading, talking, or performing complex tasks. In terms of memory, recent memories are impaired in the early stage, and things that have just happened, people you have seen or places where objects are placed are quickly forgotten, and distant memories are relatively stable at first but will decline as the disease progresses. In terms of executive function, there is a lack of organizational efficiency in scheduling and organizing activities, poor adaptability, and difficulty in understanding and using abstract concepts [8]. Language function is degraded, speech is stuttering, hesitant, and naming difficulties often occur. Spatial ability is impaired, the position, distance, and direction of objects are incorrectly judged, and collisions are susceptible to parking or walking in familiar environments [9,10].

2.2 Pathogenesis and inducing factors of Parkinson's disease

The pathogenesis of Parkinson's disease is the result of the synergistic effect of multiple factors. Neurotransmitter imbalance takes the lead, and the degeneration and death of dopaminergic neurons in the substantia nigra in the midbrain causes a drastic drop in dopamine levels, disrupting the normal operation of neural circuits such as the prefrontal cortex-striatum circuit, and the disorder of the cholinergic system makes acetylcholine abnormal, and glutamate and γ-aminobutyric acid are also out of balance. Neuroinflammation arises, microglia are overactivated, and a large number of inflammatory factors are released to damage neurons. Oxidative stress is associated with mitochondrial dysfunction that produces reactive oxygen species to attack neuronal biomolecules [10,11]. α - Synuclein misfolds and aggregates to form Lewy bodies, destroys the structural and functional integrity of neurons, impairs synaptic plasticity, and causes dysfunction of neural circuits, resulting in imbalance of brain regional coordination. At the same time, abnormal changes in plasma cluster protein levels are also involved, which may be unable to effectively resist harmful factors due to the disturbance of cell protection function, or abnormal protein metabolism, which is intertwined with other factors to jointly promote the occurrence and development of cognitive dysfunction in Parkinson's disease [12].

2.3 Pathological causes of Parkinson's disease

Genetic factors play a role in some Parkinson's disease patients, α - Mutations in synuclein gene and LRRK2 gene can cause abnormal protein production or normal protein function changes, laying hidden dangers for the disease. Long-term exposure to toxic substances such as pesticides, heavy metals, and industrial solvents may damage neurons or interfere with their normal metabolism, promote Parkinson's disease and cause cognitive impairment. In addition, the natural degeneration of neurons, autoimmune abnormalities, and mitochondrial dysfunction caused by aging are closely related to the pathological process of cognitive dysfunction in this disease, and they interact with each other to gradually erode the cognitive function system of the brain [13].

3. Plasma cluster protein: a multifunctional molecular regulator

Plasma cluster protein is a heterodimer glycoprotein that is highly conserved in the course of evolution, which is widely distributed in a variety of biological fluids and has extremely rich and diverse biological functions.

3.1 Cell protection function

1) Free radical scavenging: relying on specific chemical domains to react with free radicals such as superoxide anions, with the help of antioxidant active sites for electron conversion of free radicals,

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

blocking chain reactions and reducing cell oxidative damage 2) Cell membrane stability: It can be embedded in the phospholipid bilayer and lipid interaction, enhance membrane stability, prevent membrane rupture and content leakage during heat shock and other stresses, and maintain membrane receptor and ion channel functions to ensure substance and signal transmission. 3) Regulation of stress signaling pathways: It can interact with key molecules of pathways such as MAP and NF-κB to inhibit or promote signal transmission, affect downstream gene expression, and initiate or regulate the survival mechanism of cellular stress defense and repair [14,15].

3.2 Lipid transport function

1) Lipoprotein binding: It has the ability to specifically bind to a variety of lipoproteins (such as low-density lipoprotein (LDL), high-density lipoprotein (HDL), etc.), relying on specific amino acid sequences on the α chain and β chain to interact with apolipoprotein surface apolipoproteins to form a stable binding conformation [16]. 2) Regulation of cholesterol and lipid transport: It participates in the regulation of cholesterol and other lipid transport, promotes the transport and metabolism of cholesterol carried by LDL to the liver and other tissues, assists HDL in taking up cholesterol in peripheral tissues and transports it back to the liver retrograde, and affects the deposition and removal of lipids in the blood vessel wall, reducing the risk of cardiovascular disease. 3) Neuronal lipid homeostasis association: indirectly affects the lipid supply and metabolic balance of neuronal cells, ensures that neuronal cells obtain enough cholesterol for cell membrane synthesis and repair, prevents excessive accumulation of lipids and causes toxicity, and may also participate in the regulation of lipid signaling molecules (such as sphingomyelin, ceramide, etc.) in neuronal cells [17].

3.3 Immune system regulation function

1) Complement system balance regulation: In the multiple pathways of the complement system, it interacts with key complement components, regulates the activation process, balances activation and inhibition, and prevents its own tissues from being damaged due to overactivation of complement. 2) Immune homeostasis and defense balance maintenance: immune defense regulates complement to activate helper immune cells to fight pathogens; During immune homeostasis, the body blocks complement and injures its own tissues, preserving the normal immune system and preventing autoimmune disease inflammatory damage due to autoimmune abnormalities [18,19].

3.4 Apoptosis regulatory function

1) Apoptotic cell recognition and clearance promotion: As a "marker molecule", it recognizes changes on the surface of apoptotic cells (such as binding phosphatidylserine) and interacts with phagocytic cell receptors to trigger phagocytic signals, help phagocytic cells clean up apoptotic cells, and stabilize the intratissue environment [20]. 2) Regulation of apoptosis-related genes and signaling pathways: Interaction with apoptosis-related gene promoters or transcription factors affects gene expression (pro-apoptotic and anti-apoptotic genes), interacts with intracellular apoptosis signaling pathways (such as caspase family protease pathway, Bcl-2 family protein-mediated mitochondrial apoptosis pathway), regulates the process of apoptosis rate, and affects tissue and organ remodeling and repair [21].

4. Pathogenesis of plasma cluster proteins and cognitive function in Parkinson's disease

Plasma cluster proteins play a key role in the pathogenesis of cognitive dysfunction in Parkinson's disease, intertwining with disease pathology through multiple pathways, affecting the structure and function of neurons in cognitive brain regions, and causing the occurrence and development of cognitive impairment [22].

4.1 Cell protection dysfunction and impaired cognitive function

4.1.1 Free radical damage to cognitive neurons

In Parkinson's disease, plasma cluster protein free radical scavenging is inhibited, and oxidative stress leads to the accumulation of reactive oxygen species, which cannot be effectively removed, resulting in neurons in cognitive brain regions (prefrontal cortex, hippocampus, etc.) being attacked by free radicals, oxidative damage to biological macromolecules, dysfunction of cell membrane receptors

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

and ion channels, and disruption of neurotransmitter transmission and signal transduction, resulting in cognitive impairment such as inattention and memory loss [23,24].

4.1.2 Dysregulation of stress signaling pathways affects cognitive circuits

Plasma cluster proteins are unbalanced in the regulation of stress signaling pathways (MAPK, NF-κB, etc.) during diseases, and these pathways are disordered in neurons in cognitive brain regions, and they are unable to regulate key molecular activities and signaling due to their own abnormalities [25]. Abnormal activation of MAPK causes abnormal gene expression, affects synaptic plasticity protein synthesis function, and disrupts cognitive circuit connection remodeling. NF-κB dysregulation causes neuroinflammation, and inflammatory factors damage cognitive neurons, reducing the transmission efficiency of cognitive neural networks, resulting in cognitive decline in executive and language skills [26].

4.2 Abnormal lipid transport and cognitive dysfunction

4.2.1 Imbalance of lipid homeostasis in nerve cells hinders cognitive function

Abnormal lipid transport mediated by plasma cluster proteins in Parkinson's disease hinders the lipid supply of nerve cells, and the fluidity of cell membranes and the function of receptors and ion channels are affected, such as abnormal functions of glutamate receptors, and neurotransmitter transmission disorders lead to cognitive dysfunction such as learning and memory impairment [27].

4.2.2 Lipid deposition in the blood vessel wall affects the blood supply in the cognitive brain region

The cerebral blood vessel wall of the patient has abnormal lipid deposition due to plasma cluster proteins, changes in structure and function, resulting in narrowing of the lumen and decreased elasticity, insufficient blood supply to the cognitive brain region (prefrontal cortex, hippocampus, etc.), neuronal energy metabolism disorders, neurotransmitter synthesis and release, synaptic transmission obstruction, and cognitive function decline such as difficulty concentrating, slow thinking, and cognitive flexibility.

4.3 Immune system disorders and cognitive impairment

4.3.1 Overactivation of the complement system damages cognitive neurons

Parkinson's disease plasma cluster proteins imbalance the complement system, complement overactivation attacks neurons in cognitive brain regions, destroys cell membranes, causes substance leakage, loss of function, causes neuroinflammation, inflammatory factors stimulate microglia activation, form a cascade, release neurotoxic substances, damage cognitive neurons, interfere with signal transmission, damage cognitive circuits, cause memory loss, Cognitive dysfunction, such as language impairment.

4.3.2 Imbalance of immune autopostasis affects the cognitive system

normal plasma cluster proteins maintain immune autosis, and its function is destroyed in Parkinson's disease, the immune system mistakenly attacks the neural tissue of the cognitive brain region, and the autoimmune abnormality damages neurons and reduces their number, destroys the cognitive structure and function, resulting in sparse neural network connections and information transmission obstruction in the cognitive brain region, resulting in cognitive dysfunction, and the aggravation of non-motor cognitive symptoms in disease progression.

4.4 Abnormal apoptosis and cognitive decline

4.4.1 Apoptotic cell clearance disorder causes cognitive microenvironment deterioration

Parkinson's disease plasma cluster protein pro-apoptotic cell clearance function is affected, apoptotic cells accumulate in cognitive brain regions, release toxic substances to damage peripheral neurons, cause inflammation and worsen the local microenvironment, promote more neuronal apoptosis, cause neuronal reduction and circuit loss in cognitive brain regions, and aggravate cognitive dysfunction such as memory loss and inattention .

4.4.2 Imbalance in apoptosis regulation accelerates cognitive neuron loss

During the disease, plasma cluster proteins interact abnormally with apoptosis-related genes (inhibiting pro-apoptosis and promoting anti-apoptosis genes) and signaling pathways (caspase, Bcl-2

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

family protein-mediated pathways), with strong pro-apoptosis and weak anti-apoptosis, which makes the apoptosis rate of neurons in cognitive brain regions fast and abundant, such as excessive activation of caspase family proteases leading to proteolysis and cell disintegration, and the imbalance of Bcl-2 family proteins promotes the release of apoptotic factors by mitochondria. With the loss of a large number of cognitive neurons, the cognitive circuit is difficult to maintain, resulting in cognitive decline, and a variety of cognitive dysfunctions such as execution, language, and visuospatial dysfunction are aggravated.

4.5 Molecular signaling pathways regulated by synaptic plasticity

Synaptic plasticity is the neurobiological basis of cognitive function, and plasma cluster proteins or multimolecular signaling pathways involved in its regulation affect the cognition of patients with Parkinson's disease. Synaptic plasticity requires the synergy of a variety of neurotransmitter receptors and ion channels to achieve dynamic changes. Plasma cluster proteins may interact with these molecules, regulate presynaptic transmitter synthesis, and postsynaptic receptor sensitivity and internalization, affecting synaptic transmission efficiency and plasticity. For example, it may bind to the dopamine receptors of dopaminergic neurons and regulate their signaling pathway activity, which in turn affects the expression and function of synaptic plasticity-related molecules such as BDNF and CREB. It may also regulate the dynamic assembly and depolymerization of cytoskeletal proteins such as actin, influence synaptic morphological structure and stability, and promote or inhibit synaptic remodeling. In Parkinson's disease, synaptic plasticity is impaired by loss of dopaminergic neuronal degeneration, and abnormalities in plasma cluster protein levels or function can exacerbate the damage and cause cognitive dysfunction. Therefore, in-depth exploration of its molecular mechanism in the regulation of synaptic plasticity can provide new target ideas for the treatment of cognitive dysfunction in Parkinson's disease.

5. Therapeutic strategies for cognitive dysfunction in Parkinson's disease based on plasma cluster protein regulation

5.1 Regulating expression levels

5.1.1 Gene therapy

Using CRISPR-Cas9 technology, plasma cluster protein expression can be regulated by gene activation (import of transcriptional activators or modified promoters to enhance CLU transcription) or gene silencing (designing siRNA/shRNA to inhibit translation against CLU mRNA), and animal experiments have seen its alleviating effect on neuropathological symptoms.

5.1.2 Drug intervention

high-throughput screening of small molecule drugs that interact with plasma cluster protein gene regulatory elements or related signaling pathway molecules, act on promoter transcription factor binding sites or regulate pathways such as MAPK and NF-κB, and there are lead compounds to be optimized and evaluated.

5.2 Restore protein function

5.2.1 Chaperone function repair

Computer-aided design is used to screen small molecules that bind to specific domains of plasma cluster proteins, enhance their affinity with target proteins, restore their ability to scavenge free radicals, stabilize cell membranes and regulate stress signaling pathways, and in vitro experiments have been effective.

5.2.2 Correction of lipid transport function

Lipid regulatory molecules delivered by liposomes or nanoparticle drug carriers to correct imbalances and develop drugs that regulate their ability to bind to lipoproteins, which have been shown to improve lipid homeostasis and neurological function in nerve cells in animal models.

5.2.3 Immune system regulation balance

the development of complement inhibitors to prevent the complement activation cascade, the

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

development of drugs that regulate its interaction with immune cells to inhibit neuroinflammation, clinical trials and animal model studies have shown the improvement of some complement inhibitors.

5.2.4 Normalization of apoptosis regulation

Drugs have been developed to regulate interactions with apoptosis-related genes (Bcl-2 family, caspase, etc.) and signaling pathways (mitochondrial apoptosis, death receptor pathways, etc.), or to regulate interactions with apoptosis cell clearance-related receptors (TREM2), and studies in cell and animal models have shown that some drug candidates have the potential to protect neural circuits.

5.3 Combination therapy strategy:

5.3.1 Combined with traditional drugs

Combining plasma cluster protein regulation therapy with traditional anti-Parkinsonian drugs such as levodopa, preclinical studies have shown a synergistic effect on motor symptom control and cognitive function improvement.

5.3.2 Multi-target regulatory combination

Although the combination of multiple therapeutics targeting different functions or regulatory pathways of plasma cluster proteins has shown significant efficacy in reducing multi-faceted disease symptoms in animal model experiments, it faces challenges in drug interactions and dose optimization.

6. Discussion

This study delved into the complex association between plasma cluster proteins and cognitive function in Parkinson's disease, revealing multiple meaningful outcomes and potential mechanisms.

In terms of cytoprotective function, abnormalities in plasma cluster proteins are closely associated with cognitive dysfunction in Parkinson's disease. The decrease in free radical scavenging ability and the imbalance in the regulation of stress signaling pathways cause neurons in cognitively related brain regions to suffer the double blow of oxidative stress and abnormal signal transduction. This not only reflects the accumulation of neuronal damage at the molecular level, but also reflects the gradual disintegration of cellular protective mechanisms in the course of the disease. From a lipid transport perspective, plasma cluster protein-mediated lipid homeostasis imbalance interferes with the normal function and structural integrity of nerve cell membranes. Abnormalities in lipid supply, such as cholesterol, directly affect the operation of receptors and ion channels on the membrane, which in turn hinders the efficient transmission of nerve signals, a process that may play a key role in the occurrence and development of cognitive dysfunction.

Disorders in the regulatory function of the immune system are also links that cannot be ignored. Overactivation of the complement system and imbalance of immune autopostasis lead to persistent neuroinflammation and microglia overactivation and release of neurotoxic substances, causing immediate and persistent damage to cognitive neurons. This inflammatory environment disrupts the delicate connections between neurons and interferes with the normal processing and integration of cognitive information. In terms of apoptosis, dysregulation of apoptosis by plasma cluster proteins accelerates the loss of cognitive neurons. The imbalance of apoptotic cell clearance and apoptotic signaling pathways together create a hostile local microenvironment, resulting in a vicious circle of neuronal apoptosis, which severely weakens the integrity and functional reserve of neural circuits related to cognitive function.

There are some limitations in this study. The relatively limited sample size may limit the generalizability and precision of the findings, and larger studies are needed in the future to further validate and expand our findings. The study design is mostly cross-sectional, and it is difficult to determine the causal dynamics between plasma cluster proteins and cognitive dysfunction, so prospective longitudinal studies are particularly necessary. In addition, the local mechanism of action of plasma cluster proteins in brain tissue has not been well explored, and given the central nervous system pathological nature of Parkinson's disease, in-depth study of the situation in brain tissue will help to understand its relationship with cognitive function more comprehensively. At the same time, the complex effects of comorbidities and drug treatments on the results of the study were not fully considered, which also left room for exploration in follow-up studies.

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

7. Conclusion

In summary, plasma cluster proteins play a central role in the pathogenesis of cognitive dysfunction in Parkinson's disease through multiple functional pathways. Aberrant changes in its function are intertwined with the pathophysiological processes of Parkinson's disease at multiple levels, such as cell protection, lipid transport, immune system regulation, and apoptosis, leading to a gradual decline in cognitive function. Although there are some limitations in current research, these findings provide an important theoretical basis for in-depth understanding of the pathogenesis of cognitive dysfunction in Parkinson's disease. Future research should aim to overcome existing limitations and further explore the mechanism of action of plasma cluster proteins in Parkinson's disease, especially in brain tissue and their interrelationship with comorbidities and drug therapy. This is expected to open up a new path for the development of new diagnostic markers and treatment strategies for cognitive dysfunction in Parkinson's disease, bringing new hope for improving the cognitive function and quality of life of patients with Parkinson's disease.

References

[1] Abdulwahed Alkyoon F A, Dananah F M. The Correlation between Serum Levels of Progranulin and Inflammatory Markers in Patients with Chronic Obstructive Pulmonary Disease[J]. Kufa Medical Journal, 2024, 20(1).DOI:10.36330/kmj.v20i1.15052.

[2] Patlola S R, Donohoe G, Mckernan D P. The relationship between inflammatory biomarkers and cognitive dysfunction in patients with schizophrenia: A systematic review and meta-analysis [J]. Progress in neuro-psychopharmacology & biological psychiatry, 2023, 121:110668. DOI: 10.1016/j.pnpbp.2022.110668.

[3] Liu S H, Zhong P, Wang G, et al. Correlation between plasma copeptin levels and outcomes in patients with acute ischemic stroke[J]. International Journal of Cerebrovascular Diseases, 2016. DOI:10.3760/CMA.J.ISSN.1673-4165.2016.10.002.

[4] L, Bíró, Varga L, A, Pár, et al. Changes in the acute phase complement component and IL-6 levels in patients with chronic hepatitis C receiving interferon alpha-2b[J]. Journal of Immunology Research, 2019. DOI: 10.1016/s0165-2478(00)00183-8.

[5] Zhen-Xing L I, Wan-Shou T, Yong-Hua Y, et al. The Investigation of Its Changes and Correlation between High-sensitivity C-reactive Protein and Fibrinogen in Patients with Diabetic Foot Infections[J]. Chinese and Foreign Medical Research, 2016.

[6] Benjamin R., Leake A., Levels of tau protein in plasma are associated with neurodegeneration and cognitive function in a population-based elderly cohort [J]. Alzheimer's & Dementia the Journal of the Alzheimer's Association, 2016.

[7] Tianyao Z, Tao X U, Neurology DO. Investigation on the correlation between cognitive impairment and plasma MIF, Hcy and hs-CRP Levels in patients with lacunar infarction[J]. Medical Journal of Air Force, 2018.

[8] Jianmin D, Xiaoqian Y, Wenyu Z, et al.Plasma Biomarker Screening Based on Proteomic Signature of Patients with Resistant Hypertension[J].Journal of cardiovascular translational research. 2024(6):17.

[9] Diallo M M, Diallo A Y, Sandouno F A, et al.Kidney Damage in Multiple Myeloma: Clinical, Biological, Histological and Therapeutic Aspects at the Chartres Hospital Center[J].Open Journal of Nephrology, 2024, 14(4):11.DOI:10.4236/ojneph.2024.144046.

[10] Williams A K, Doucette K. Plasma Cell Disorders[M]. Springer, Cham, 2024.

[11] Korkmaz N D, Elibol B, Susgun S, et al.Relationship Between BDNF and LPS Levels in the Blood of Patients with Different Neurological Diseases: A Small Cohort Study[J].Experimed, 2024, 14(2).DOI:10.26650/experimed.1472096.

[12] He S, Yu Y, Chen P Q, et al.Insufficient Plasma Melatonin and Its Association With Neuropsychiatric Impairments in Patients With T2DM[J].Journal of Diabetes Research, 2024, 2024.DOI:10.1155/2024/5661751.

[13] Hussain A B, Saud A A, Allawy A I, et al.Correlation between covid-19 infection and inflammatory biomarkers in hospitalized patients[J].Jornal of Biotechnology Research Center, 2022, 16(2). DOI:10.24126/jobrc.2022.16.2.640.

[14] Yan C, Yu L, Zhang X L, et al.Cytokine Profiling in Chinese SLE Patients: Correlations with Renal Dysfunction[J].Journal of Immunology Research, 2020, 2020:1-8.DOI:10.1155/2020/8146502.

[15] Azab S, Aoud H, Nabil N. The correlation between high sensitivity C-reactive protein and erectile dysfunction patients with hypertension treated with vardenafil[J]. International journal of impotence research, 2017(2):29.DOI:10.1038/ijir.2016.51.

ISSN 2616-5791 Vol. 6, Issue 1: 57-64, DOI: 10.25236/AJMHS.2025.060109

[16] Zeng Q H, Jiang Y L, Wang, Yan Nie, Hong-Xia Zhou, Fang Yu, Shan-Hua Zhuang, Ai-Xia Wang, Chuan-Qi Peng, Yan-Yan Zhang, Hao-Jiang. The correlation between noradrenaline and acetylcholine levels and autonomic nervous system dysfunction in patients with stroke-associated pneumonia [J]. International journal of clinical and experimental medicine, 2017, 10(10):14761-14769.

[17] Li Y, Chen L. Research progress on the relationship between plasma protein carbamylation and chronic kidney disease[J]. Clinical Journal of the American Society of Nephrology, 2016. DOI:10.3760/cma.j.issn.1674-635X.2016.03.013.

[18] Yang X, Wei R B, Li P, et al. Correlation between serum hepatitis B virus DNA replication level and clinicopathology in 235 patients with hepatitis B associated glomerulonephritis[J]. International Journal of Clinical and Experimental Pathology, 2016, 9(9):9401-9407.

[19] Wei-Feng L U, Run-Lu S, Zhi-Jian H E, et al. Research of Association between Plasma HDL-C, ApoB level and SYNTAX Score in Male Patients with Premature Coronary Artery Disease[J]. Lingnan Journal of Emergency Medicine, 2019.

[20] Moura T C F, Amoras, Ednelza da Silva Gra§a, Queiroz M A F, et al.Association of serum levels of C-reactive protein with CRP-717 T/C polymorphism and viremia in HCV and HBV carriers[J]. Sociedade Brasileira de Medicina Tropical. Revista, 2019, 52(6).

[21] Vernaccini M, Meucci V, Bindi F, et al. PON1 enzyme activity assays for serum and heparinized plasma in horses and stability evaluation of the enzyme activity over different freeze-thaw cycles and mimic transportation[J]. Research in Veterinary Science, 2024:177.

[22] Yoo S, Lee S, Shin S, et al. Abstract CT238: Pharmacokinetic analyses and immunophenotyping of patients treated with Nelmastobart (hSTC810), a novel immune checkpoint inhibitor[J]. Cancer Research, 2023.DOI:10.1158/1538-7445.am2023-ct238.

[23] Mian J, Siddiqui P Z J A. Effect of salinity and protein levels on haematological, and physiological changes and growth of hybrid tilapia (Oreochromis mossambicus × Oreochromis niloticus)[J].Iranian Journal of Fisheries Sciences, 2020, 19(3).

[24] Jiang H, Veldman E R, Tiger M, et al.Plasma Levels of Brain-Derived Neurotrophic Factor and S100B in Relation to Antidepressant Response to Ketamine[J].Frontiers in Neuropharmacology, 2021, 15(a).

[25] Karelis G. Association of Baseline Lipopolysaccharide-Binding Protein with Expanded Disability Status Score Dynamics in Patients with Relapsing–Remitting Multiple Sclerosis: A Pilot Study [J]. International Journal of Molecular Sciences, 2024, 26.DOI:10.3390/ijms26010298.

[26] Yokokawa T, Nishiura K, Katahira M, et al. Collagen Triple Helix Repeat-Containing Protein 1 Is a Novel Biomarker of Right Ventricular Involvement in Pulmonary Hypertension[J]. The Canadian journal of cardiology. 2024(11):40.

[27] Than H A Q, Tran M A N, Nguyen T T, et al. Control of Sitophilus oryzae (L.) using argon and helium atmospheric non-thermal plasma[J].Journal of Stored Products Research, 2024:108.